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FOOD-PAIRED STIMULI AS CONDITIONED REINFORCERS: EFFECTS OF d-AMPHETAMINE

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Seven pigeons were studied in two experiments in which key pecks were reinforced under a second-order schedule wherein satisfaction of variable-interval schedule requirements produced food or a brief stimulus. In the second part of each session, responses produced only the brief stimulus according to a variable-interval schedule (food extinction). For the 4 pigeons in Experiment 1, the response key was red throughout the session. In separate phases, the brief stimulus was either paired with food, not paired with food, or not presented during extinction. *d*-Amphetamine (0.3 to 10.0 mg/kg) dose-dependently reduced food-maintained responding during the first part of the session and, at intermediate dosages, increased responding during the extinction portion of the session. The magnitude of these increases, however, did not consistently depend on whether the brief stimulus was paired, not paired, or not presented. It was also true that under nondrug conditions, response rates during extinction did not differ reliably depending on pairing operations for the brief stimulus. In Experiment 2, 3 different pigeons responded under a procedure wherein the key was red in the component with food presentations and blue in the extinction component (i.e., multiple schedule). Again, *d*-amphetamine produced dose-related decreases in responding during the first part of a session and increases in responding in the second part of the session. These increases, however, were related to the pairing operations; larger increases were observed when the brief stimulus was paired with food than when it was not or when it was not presented at all. Under nondrug conditions, the paired brief stimulus controlled higher response rates during extinction than did a nonpaired stimulus or no stimulus. These findings suggest that *d*-amphetamine can enhance the efficacy of conditioned reinforcers, and that this effect may be more robust if conditioned reinforcers occur in the context of a signaled period of extinction.

Key words: conditioned reinforcement, *d*-amphetamine, brief stimulus, second-order schedules, key peck, pigeons

Stimuli associated with reinforcers may serve reinforcing functions themselves, and such stimuli are called conditioned reinforcers (Mazur, 1990; Skinner, 1938). It has been suggested that drugs clinically classified as stimulants may enhance the conditioned reinforcing effects of stimuli (e.g., Hill, 1970; T. Thompson, 1984). In several studies, stimulants have increased response rate during extinction when responding resulted in the brief presentation of stimuli that were associated with unconditioned reinforcers (e.g., Beninger, Hanson, & Phillips, 1980, 1981; Beninger & Phillips, 1980; Hill, 1970; Hoffman & Beninger, 1985; Mason & Robbins, 1979; Mazurski & Beninger, 1986; Robbins, 1978; Robbins & Koob, 1978; Taylor & Robbins, 1984). For example, Hill (1970) reinforced rats' responding with milk under a variable-interval (VI) schedule of reinforcement. These sessions were followed by an extinction session in which milk was no longer presented, but responses for some rats produced the sound of the milk dispenser (i.e., presumed conditioned reinforcement). When lever pressing produced the sound, rats given pipradrol showed enhanced response rate compared to rats given placebo injections or rats given pipradrol to whom the sound was not presented.

As in the study by Hill (1970), most research investigating the relationship between conditioned reinforcement and stimulants has employed extinction procedures and between-group designs (e.g., Mazurski & Beninger, 1986; Robbins, 1978). Under extinction procedures, a neutral stimulus is first associated with an unconditioned reinforcer, and then its

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reinforcing effects are measured in the absence of the unconditioned reinforcer. These techniques generally were abandoned several years ago by those studying conditioned reinforcement because of difficulties in interpreting the results in terms of reinforcement and because of the often weak and transient effects of the putative conditioned reinforcer (see Hendry, 1969; Kelleher & Gollub, 1962; Wike, 1966).

Extinction techniques were replaced by chained-schedule and brief-stimulus procedures (see Fantino, 1977; Gollub, 1977; Kelleher, 1966; Marr, 1969). Under brief-stimulus procedures (e.g., second-order schedules), responding is maintained by the intermittent presentation of unconditioned reinforcers, while responding also results in the brief presentation of conditioned reinforcers (e.g., Stubbs, 1971). Although several experiments have examined the effects of psychomotor stimulants on second-order schedule performance (e.g., Barrett, Katz, & Glowa, 1981; Bond, Sanger, & Blackman, 1975), there is relatively little research available involving other brief-stimulus procedures (see Herling, Downs, & Woods, 1979; Kelly & Thompson, 1985).

Recently, Files, Branch, and Clody (1989) examined the effects of methylphenidate in a novel, within-subject procedure designed to examine the effects of conditioned and unconditioned reinforcement. Under this procedure, brief stimuli served as putative conditioned reinforcers during extinction. Pigeons' responses in the first part of each session produced food according to a random-ratio (RR) 2 (VI 30 s: S^P) second-order schedule. Under this schedule, a response after an average of 30 s produced a 3-s brief stimulus (S^P, houselight off, key color change, tone, and hopper light) and, following completion of two VI schedule requirements on average, a response produced the brief stimulus accompanied by food delivery. Following 10 to 20 food presentations, a 20-min extinction period was initiated that was not signaled by any stimulus change (i.e., the procedure was technically a mixed schedule of reinforcement). In this component food was never presented; rather, in some sessions responses produced the brief stimulus according to a VI 30-s schedule, and in other sessions responses had no scheduled consequences. Methylphenidate produced higher response rates in the extinction component when responding produced a brief stimulus compared to when the stimulus was not presented.

Although methylphenidate enhanced responding when brief stimuli were presented, an interpretation of the data in terms of conditioned reinforcement remains equivocal. Files et al. (1989) used only a food-paired brief stimulus. A conditioned reinforcement interpretation would be strengthened if the stimulant was shown to have less of a rate-increasing effect with a nonpaired brief stimulus. Perhaps stimulants enhance the reinforcing sensory effects of response-dependent brief stimuli (cf. Kish, 1966), whether or not they are paired with food. Interestingly, Files et al. (1989) also failed to observe a clear conditioned reinforcement effect under nondrug baseline conditions; response rates in the extinction component were similar during sessions with and without a brief stimulus. Conditioned reinforcement would have been in evidence had response rate been greater when the brief stimulus was available.

The present study attempted to extend the findings of Files et al. (1989). In Experiment 1, a procedure was used that closely resembled the schedule used in that study, but here *d*-amphetamine was administered under conditions in which a brief stimulus was either paired or not paired with food. In Experiment 2, the two components (food and extinction) were signaled by different key colors (i.e., a multiple schedule).

EXPERIMENT 1

Method

Subjects

Four experimentally naive male White Carneau pigeons (Palmetto Pigeon Plant) were maintained at 80% of their free-feeding weights (403 to 497 g). Water was freely available in their home cages, where a 12:12 hr light/dark cycle was maintained (lights on at 6:00 a.m.).

Apparatus

One noncommercial and three modular (Coulbourn Instruments) pigeon chambers were used. In the modular chambers, the key (2.5 cm diameter) was located in the center of the work panel, 6.0 cm from the ceiling, and was transilluminated red, white, or blue by an

IEE one-plane readout. A minimum force of 0.10 N operated the key. A 28-V white houselight was located above the key. Mixed grain was presented through an aperture below the key and was illuminated red during each food cycle. The noncommercial chamber (see Cohen & Lentz, 1976) had a similar configuration except that a Gerbrands key (1.9 cm diameter) was located to the left of center, 4.5 cm from the ceiling, and could be operated with a minimum force of 0.07 N. Also, a Lehigh Valley Electronics pigeon feeder was used. White noise was continuously present to mask extraneous sounds. Contingencies were controlled by an IBM-PC® computer, Coulbourn Instruments Lab-Linc® Interface, and Pascal programming.

Procedure

Responding on the red key was established by the method of successive approximation. Responding was maintained by a continuous reinforcement schedule for four sessions, a VI 5-s schedule for one session, a VI 10-s schedule for two sessions, a VI 20-s schedule for one session, and a VI 30-s schedule for six sessions. Under each VI schedule, each reinforced response operated the food magazine and red feeder light for 4 s while the keylight remained red. Every VI schedule used in this experiment contained 20 intervals that were derived from the formula of Catania and Reynolds (1968, p. 380). One interval was randomly chosen following each food presentation until the entire set of 20 intervals was exhausted, at which time random selection began anew. Session duration was 30 min, and sessions were conducted Monday through Friday.

Next, a second-order schedule was initiated in which the completion of half of the VI 30-s requirements (randomly determined) produced food, and VI completions not producing food resulted in the presentation of a nonpaired brief stimulus (i.e., RR 2 [VI 30 s: S^{NP}]). The brief stimulus consisted of a 4-s change in key color from red to white plus the illumination of the white houselight. Responses during the brief stimulus had no scheduled consequences. Session time was randomly determined each day and averaged 35 min, with a range of 30 to 40 min. This condition was in effect for 24 to 28 sessions.

Nonpaired stimulus condition. Each session started with the RR 2 (VI 30 s: S^{NP}) schedule of reinforcement (Component 1). After an average of 35 min (range, 30 to 40 min), the schedule changed to extinction but the key color remained red (Component 2). Component 2 remained in effect for 30 min, and the session terminated. In Component 2, responding never produced food but instead produced the brief stimulus (4-s white keylight plus white houselight) according to a VI 30-s schedule. This condition was in effect for 35 to 47 sessions before responding became stable (no increasing or decreasing trends in response rate for at least five sessions) and the first injection was administered. Independent VI schedules were used for Components 1 and 2.

Paired stimulus condition. The brief stimulus in Component 1 was now paired with each food presentation (i.e., RR 2 [VI 30 s: S^{P}]): Otherwise, all conditions were identical to the nonpaired condition. A preceding overlapping pairing operation was used (Stubbs & Cohen, 1972), in which the completion of each VI requirement scheduled to produce food turned on the white keylight and houselight shortly before (see below) food delivery and remained on during the 4-s food cycle. Several lengths of preceding intervals were used to ascertain an optimal value: 1.0 s (9 to 24 sessions), 0.5 s (six sessions), 1.0 s (six sessions), 1.5 s (five sessions), and 1 s (7 to 10 sessions). Because no differences in response rate were observed as a function of these time intervals, the 1-s preceding interval was used during drug administration.

Nonpaired stimulus condition (replication). The first condition was replicated. Twenty-two to 28 sessions were conducted before injections were administered.

No-stimulus condition. In this phase, responding during Component 2 did not have any scheduled consequences. Otherwise, contingencies were identical to the paired brief-stimulus condition, that is, in Component 1 the brief stimulus was presented upon completion of every VI requirement and preceded (by 1 s) and accompanied each food presentation. Eighteen to 19 sessions were conducted before injections were administered.

Drug administration. *d*-Amphetamine (Sigma) was mixed in physiological saline and administered in the following doses: 0 (saline), 0.3, 1, 3, and 10 mg/kg body weight. Each

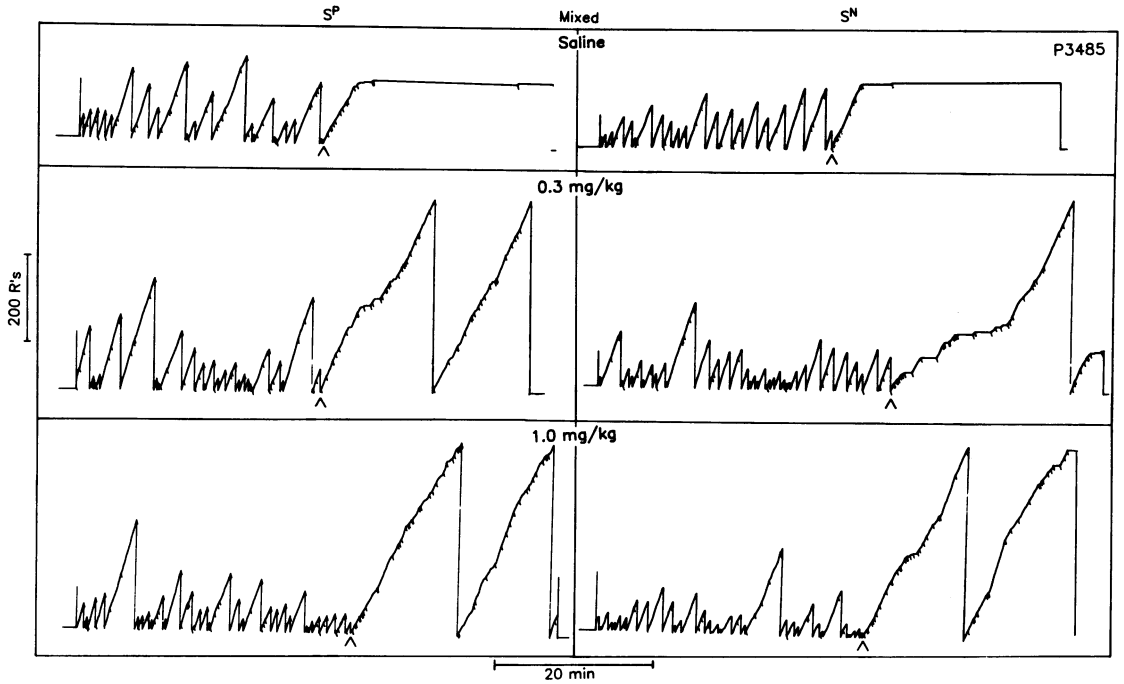

Fig. 1. Cumulative response records of key pecking by Subject P3485 under the mixed schedule (y axes, cumulative responses; x axes, time). Diagonal marks on the records indicate presentations of the brief stimulus, and the pen reset to baseline with each food delivery. The arrow indicates the beginning of the second component of the schedule: After this point, no food was presented. Records in the left column are from sessions when the brief stimulus was paired with food presentations; those in the right column are from sessions in which the brief stimulus was not paired with food presentations. Records in the top row are from sessions preceded by administration of the saline vehicle. The lower two rows contain records from sessions preceded by injection of 0.3 mg/kg (middle row) or 1.0 mg/kg (bottom row) *d*-amphetamine.

subject completed two ascending dose series. If response rate was at zero or near-zero levels at one dose, the next higher dose in that particular series was typically not administered (see figures). *d*-Amphetamine was mixed with saline in a volume of 1 mL/mg and injected in the breast muscle 10 min before the session. An injection was given Tuesday and Friday of each week.

RESULTS

Figure 1 shows cumulative response records for Subject P3485. Responding when no drug was administered occurred at a roughly constant rate when the second-order schedule was in effect. When extinction began, responding continued for a short while and then ceased. *d*-Amphetamine at 0.3 and 1.0 mg/kg produced little change in performance during the second-order schedule, but yielded large increases in responding during extinction regardless of whether the brief stimulus was paired with food presentations or was not paired.

Response rate was determined for each component separately by dividing total responses in each component by time spent in that component. Responses and time during food and brief-stimulus presentations were not included in these calculations. Figure 2 shows overall response rate in Component 1 during baseline, preinjection, and injection sessions. *d*-Amphetamine decreased response rates in a dose-dependent manner during the second-order schedule. No consistent differences between nonpaired, paired, and no-stimulus conditions were observed during drug or nondrug sessions.

Response rates during Component 2 (extinction) are presented in Figure 3. Under nondrug conditions responding was very variable from session to session and across pigeons, making comparisons between paired stimulus, nonpaired stimulus, and no-stimulus conditions difficult. There were many sessions with very few responses in Component 2. The pigeons' behavior appeared sensitive to the positive relationship between time spent in Com-

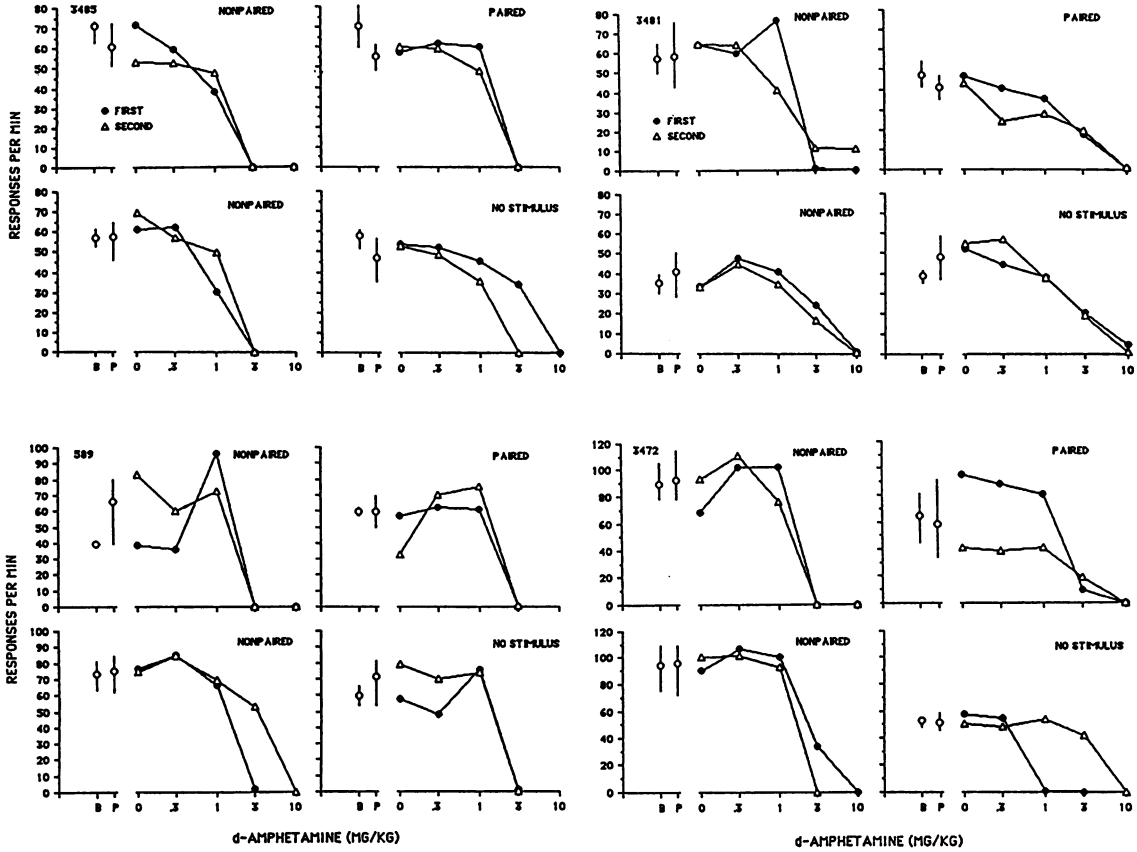

Fig. 2. Overall response rate during the second-order schedule for Conditions 1 to 4 of Experiment 1, starting with the upper left panel for each subject. Baseline (B) data show the mean and range of five sessions before the first injection. Preinjection (P) data are the mean and range of sessions before every injection. Shown separately are data from the first and second injection series. Note that the scales on the *y* axes differ among subjects.

ponent 1 and the probability of Component 2 onset (cf. Files et al., 1989), and the pigeons often stopped responding very early in the extinction component. No consistent differences were observed during baseline and preinjection sessions among nonpaired, paired, and no-stimulus conditions. In virtually every condition, small and moderate doses of *d*-amphetamine increased response rates over the mean values from baseline and preinjection sessions. Consistent differences among paired, nonpaired, and no-stimulus conditions were not observed, but there was a modest tendency for rate increases during the paired stimulus and nonpaired stimulus conditions to be greater than those in the no-stimulus condition.

The number of responses during brief-stimulus presentations were recorded. Under all conditions, less than one response on average occurred during brief-stimulus presentations, and there was no orderly relationship between drug dose and responses per brief stimulus.

EXPERIMENT 2

METHOD

Subjects and Apparatus

Three experimentally naive male White Carneau pigeons (Palmetto Pigeon Plant) were maintained at 80% of their free-feeding weights (396 to 451 g). Conditions were the same as for Experiment 1, as was the experimental apparatus.

Procedure

Pigeons received the same sequence of conditions described in Experiment 1 except that the second part of the session (extinction) was signaled by a blue keylight (multiple schedule). Subjects received 23 sessions in the preliminary second-order schedule condition. The number of sessions before drug administration was as follows: 36 sessions in the first nonpaired stimulus condition, 37 to 46 sessions in the paired stimulus condition (13 to 22 in the

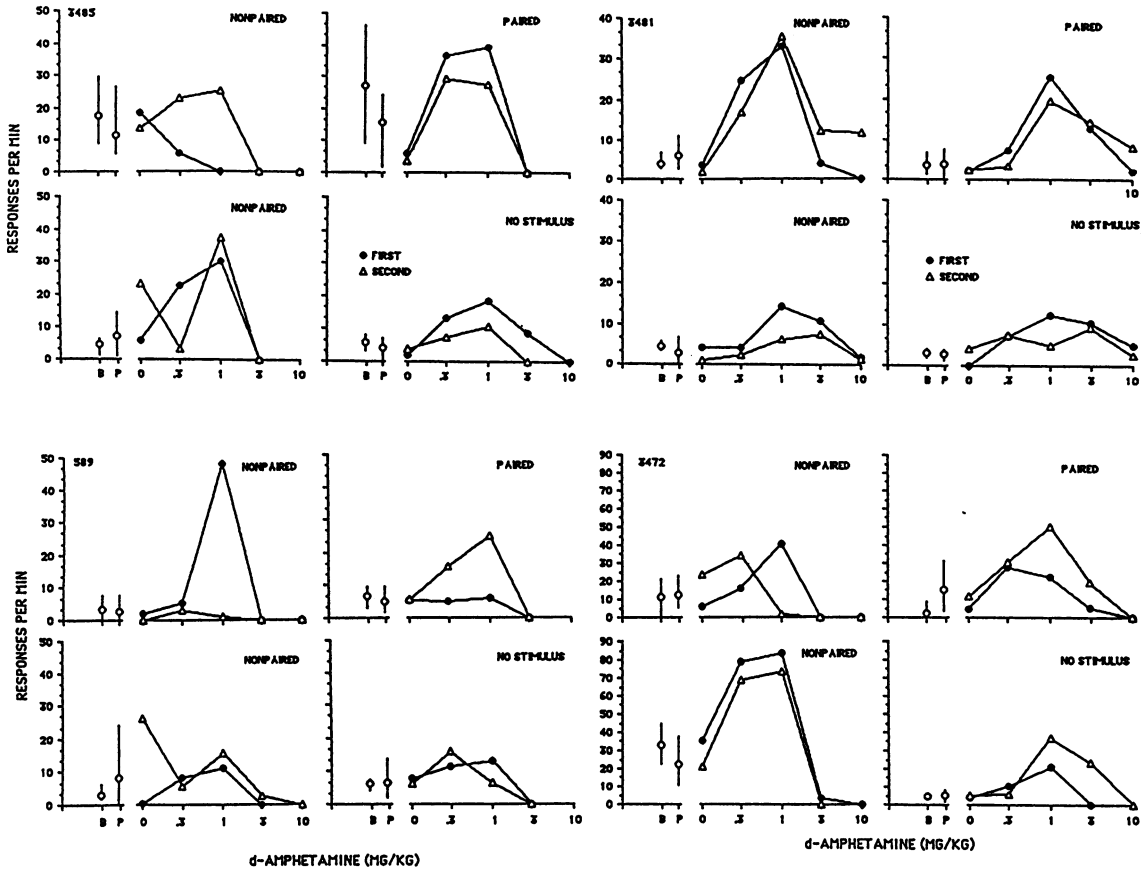

Fig. 3. Overall response rate during the extinction component for Conditions 1 to 4 of Experiment 2. Other features are the same as in Figure 2. Note that in the no-stimulus condition brief-stimulus presentations were not scheduled in the second half of the session but were paired with food under the second-order schedule.

1-s paired condition, 7 under 0.5-s pairing, 5 under 1-s pairing, 5 under 1.5-s pairing, and 7 under the last 1-s pairing operation), 19 to 30 sessions in the second nonpaired stimulus condition, and 17 to 21 sessions in the no-stimulus condition. The drug regimen was identical to that described in Experiment 1.

Results

Figure 4 displays cumulative response records for Subject P1333. Under nondrug conditions, responding occurred at a steady rate under the second-order schedule, with rate a bit more rapid when brief stimuli were not paired with food. Following the onset of extinction, responding subsided very quickly when a nonpaired stimulus occasionally followed pecks. It slowed less dramatically when a paired stimulus was an occasional consequence of pecking. *d*-Amphetamine, at 0.3 and 1.0 mg/kg, did not disrupt the constant-rate nature of performance during the second-order schedule to any great degree. Responding during extinction was increased by the drug, and the records indicate that responding occurred at a less regular rate during extinction (i.e., the records are "grainy").

Figure 5 shows overall response rate in Component 1 during baseline, preinjection, and injection sessions. Response rates were somewhat higher during nonpaired brief-stimulus conditions than during paired stimulus and no-stimulus conditions. *d*-Amphetamine decreased response rate in a dose-dependent manner during the second-order schedule.

Overall response rates during Component 2 (signaled extinction) are presented in Figure 6. A conditioned reinforcement effect can be

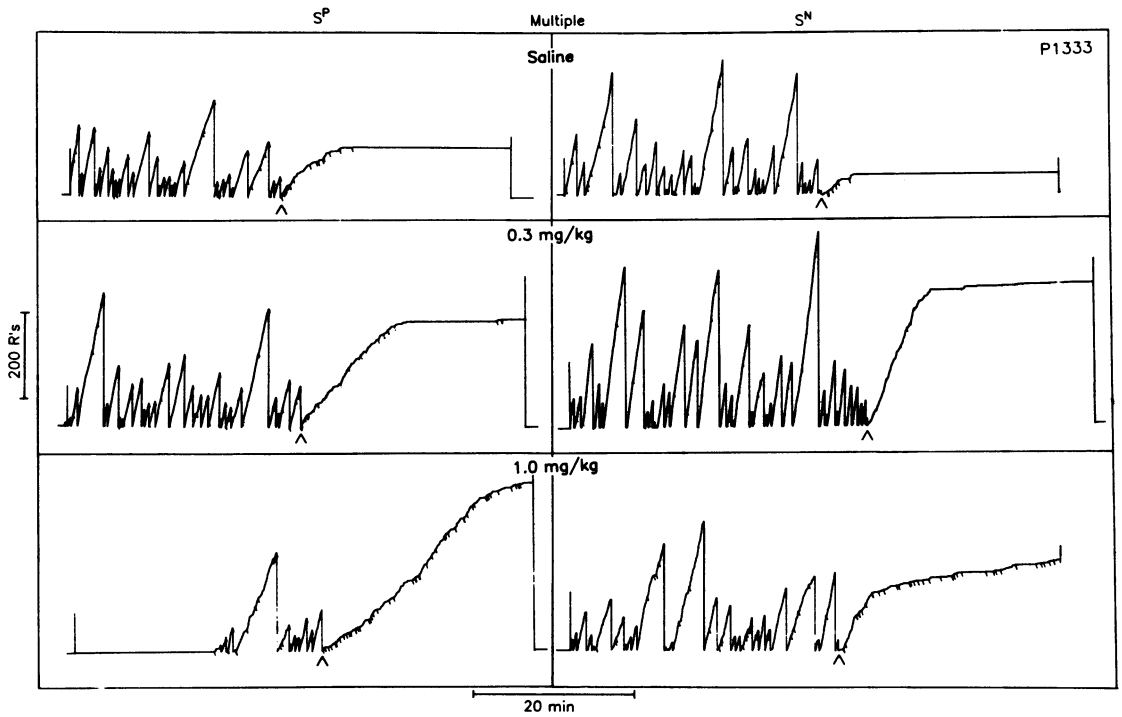

Fig. 4. Cumulative response records of key pecking by Subject P1333. Details are the same as in Figure 1.

observed under baseline and preinjection sessions. Response rate was at zero or near-zero levels under the first nonpaired stimulus condition (mean baseline and preinjection responses per minute, respectively, were 0 and 0.5 for Bird 3097, 0.1 and 2.2 for Bird 1333, and 0.2 and 0.3 for Bird 899) and increased when the brief stimulus was paired with food (0.8 and 1.5 for Bird 3097, 4.8 and 5.2 for Bird 1333, and 0.9 and 0.8 for Bird 899). Response rates decreased when the stimulus was no longer paired for Birds 3097 (0 and 0.1) and 899 (0.1 and 0.1) but remained elevated for Bird 1333 (5.8 and 4.9). When a brief stimulus was not scheduled in Component 2, responding ceased for Birds 3097 and 899 and decreased for Bird 1333 (2.6 and 1.9).

At small and moderate doses, *d*-amphetamine increased response rates over those seen in baseline and preinjection sessions. In general, enhancement of response rate during paired brief-stimulus conditions was greater than that observed during nonpaired and no-stimulus conditions. The smallest effect of *d*-amphetamine was observed during no-stimulus conditions. This relationship can be seen most clearly in the data of Birds 3097 and 1333 and to some extent in those of Bird 899.

Under all conditions, less than one response on average occurred during brief-stimulus presentations, and there was no orderly relationship between drug dose and responses per brief stimulus.

DISCUSSION

d-Amphetamine increased response rates in the extinction component in both experiments. In Experiment 1 no consistent difference was observed among conditions in which a brief stimulus was paired with food, not paired with food, or not presented at all, although there was a tendency for rates in the brief-stimulus conditions to be elevated more than those in the no-stimulus conditions. However, in Experiment 2, when extinction was signaled *d*-amphetamine produced the greatest increase in response rate with a paired brief stimulus, the smallest increase with no brief stimulus, and an intermediate effect with a nonpaired brief stimulus. That is, during signaled extinction the drug's effects depended on whether

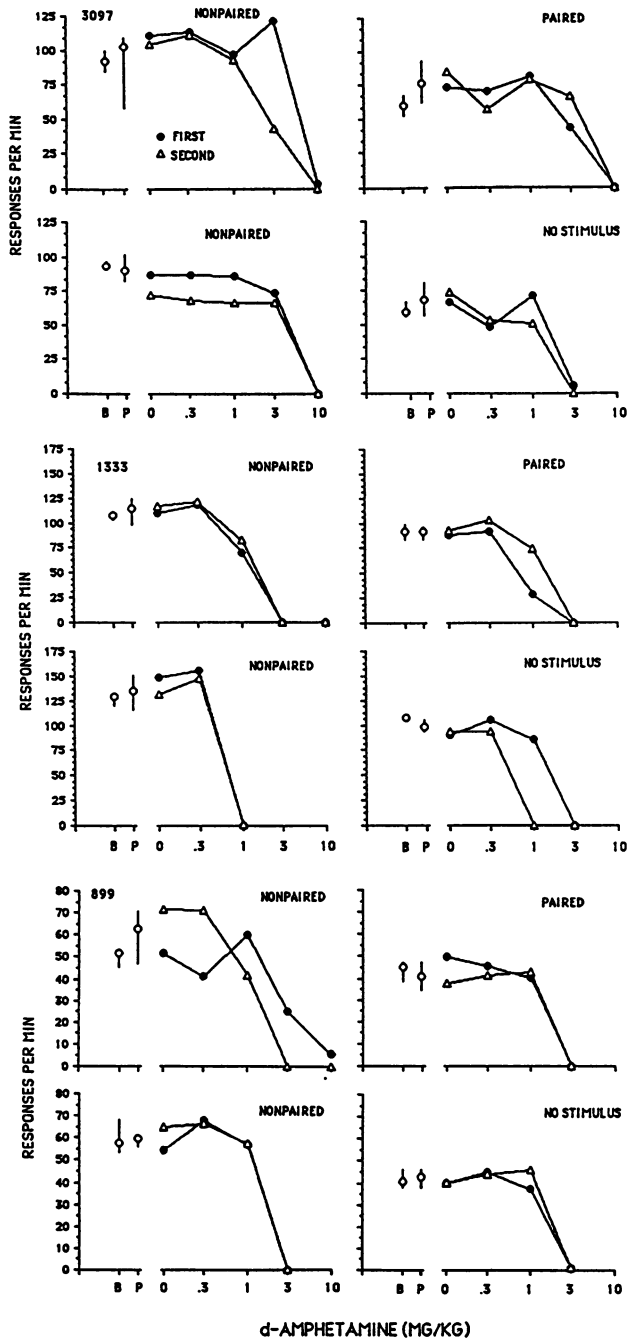

Fig. 5. Overall response rate during the second-order schedule for Conditions 1 to 4 of Experiment 2, starting with the upper left panel for each subject. Other features are the same as in Figure 2.

the brief stimulus was paired with food, whereas during unsignaled extinction reliable differences in drug effects did not emerge. In both experiments, *d*-amphetamine produced dose-dependent decreases in response rate in Component 1 (i.e., during the second-order schedule).

The data from Experiment 1 are not en-

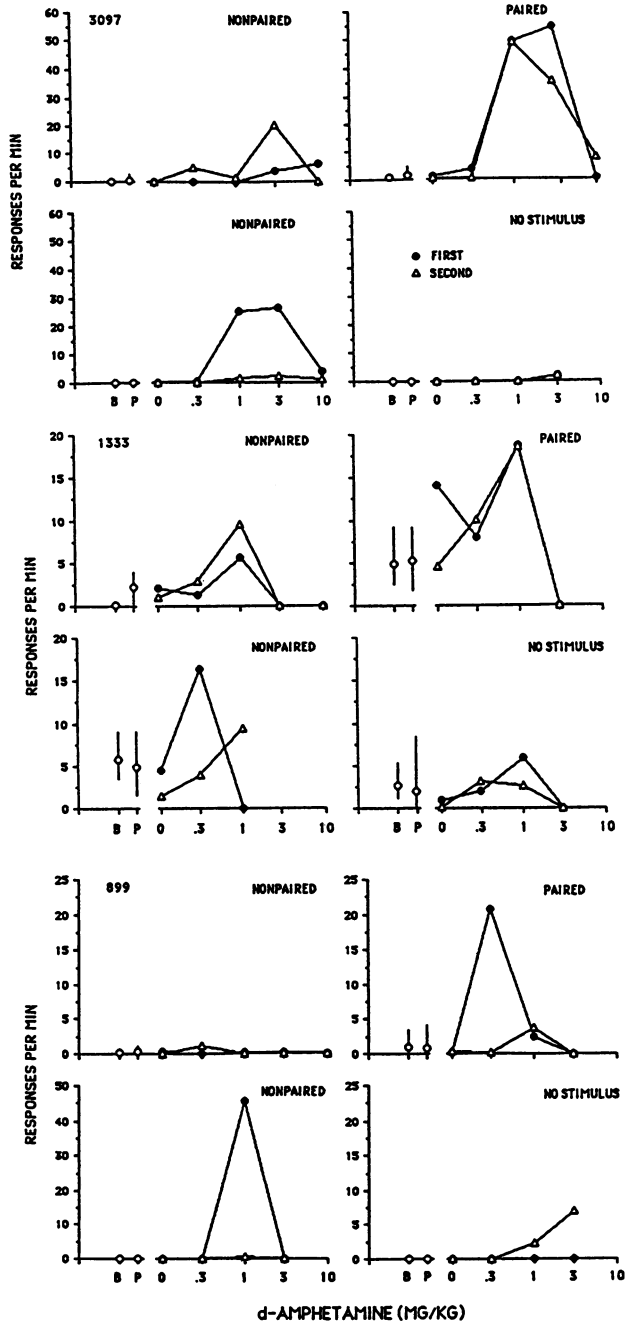

Fig. 6. Overall response rate during the extinction component for Conditions 1 to 4 of Experiment 2. Other features are the same as in Figure 3.

tirely consistent with the results of Files et al. (1989). In their experiment, responding in the extinction component was enhanced by methylphenidate during sessions with and without brief stimuli; however, greater increases were observed with brief stimuli. Although these data appear to support a conditioned reinforcement interpretation, a nonpaired brief-stimulus comparison was not included. In the present study, *d*-amphetamine also enhanced

responding in the extinction component. However, differences were not consistently observed between conditions in which stimuli were associated with food (i.e., the putative conditioned reinforcers), were not associated with food, or were not presented. In the study of Files et al., response rates during extinction increased following drug administration to a greater extent than rates in the present experiment; rates increased to levels at least as high as those that prevailed when food was available, and this was seen in all subjects. In the present study (compare Figures 2 and 3), rates were not elevated this much.

The present study and that of Files et al. (1989) differed in several procedural respects. In the Files et al. study, the first part of each session (i.e., when responding was reinforced with food presentation) was more variable in duration, the brief stimulus was more complex, whether brief stimuli were presented during extinction varied from session to session, and a different drug was employed, to name a few of the differences. Any of these may have contributed to the differences in results.

Evaluation of drug effects in Experiment 1 was made difficult by considerable session-to-session variability in response rates during the extinction components. We felt that use of a mixed schedule may have promoted this high degree of variability, so in Experiment 2 extinction was signaled. Absolute levels of baseline variability in response rate during extinction decreased, and reliable differences among the pairing conditions were observed. The differences revealed under baseline conditions indicated that, in Experiment 2, the paired brief stimulus did function as a conditioned reinforcer. Nonpaired brief stimuli did not maintain responding in the first condition. When the brief stimulus was paired with food, responding increased for all 3 subjects. When the stimulus was no longer paired, rates declined for 2 pigeons but remained high for 1. With this baseline, therefore, differences in response rate following injection of *d*-amphetamine were observed under paired and nonpaired brief-stimulus conditions: The largest increase in response rate occurred when the brief stimulus was paired with food. The data from the multiple schedule thus support the hypothesis that stimulants enhance the efficacy of conditioned reinforcers. It is not clear why *d*-amphetamine enhanced responding in the nonpaired brief-stimulus condition more than in the condition with no brief stimulus. Because the brief stimulus was not paired with food it should not have acquired reinforcing properties, although *d*-amphetamine might have affected the sensory reinforcement properties of the stimulus (Kish, 1966). Another possibility is that the nonpaired stimulus had some efficacy as a conditioned reinforcer due to generalization. Both food presentation and presentation of the nonpaired stimulus share the feature of a change in stimulation.

The data from Experiment 2 support a number of studies that used extinction procedures to demonstrate that stimulant drugs enhance the effectiveness of conditioned reinforcers. For example, Taylor and Robbins (1984) associated a brief tone, illumination of a feeder light, and offset of a houselight with presentation of water. Later, rats were given *d*-amphetamine infusions into the nucleus accumbens and placed in a chamber with two levers. With water no longer available, responding on one lever produced the brief stimulus and responding on the other lever had no effect. Rats acquired the lever-press response, and *d*-amphetamine increased responding significantly more on the lever producing the conditioned reinforcer. The present study employed a procedure in which the effects of repeated drug administrations were examined within a single subject while the conditioned reinforcer continued to be paired with the unconditioned reinforcer over successive sessions.

Although a conditioned reinforcement interpretation of the data is supported, alternative interpretations should be considered. According to the rate-dependency hypothesis, control rate of response may determine a drug's effect on response rate (e.g., Dews & Wenger, 1977; Gonzalez & Byrd, 1977). *d*-Amphetamine, for example, has been shown to increase low response rates and to decrease or not affect high response rates. In the present experiment, *d*-amphetamine lowered high rates of responding in the first half of the session (second-order schedule) and increased low rates of responding in the second half (extinction). This explanation cannot, however, account for the fact that *d*-amphetamine produced greater increases under paired brief-stimulus conditions compared to nonpaired or no-stimulus conditions, even though baseline response rates

with paired stimuli were higher than those seen in the other two conditions (see Figure 6).

It might be argued that because *d*-amphetamine can affect stimulus control (D. M. Thompson, 1978), the drug makes it difficult to discriminate between the first and second components of the multiple or mixed schedules and results in indiscriminate responding throughout the session. Components 1 and 2 would appear most alike and would present the most difficult discrimination under the paired brief-stimulus condition because a brief stimulus is presented following the completion of every VI requirement in both components. In the nonpaired condition, the brief stimulus is presented after the completion of only half of the VI requirements in the food component and all of the VI requirements in the extinction component. And, of course, the no-stimulus condition poses the easiest discrimination between components. If *d*-amphetamine interferes with the ability to discriminate among components, then more responding might be expected when the discrimination is the most difficult (i.e., the paired brief stimulus) and less responding when the discrimination is the easiest (i.e., no brief stimulus). Although it is difficult to confirm this possibility with the present procedure, it might be predicted that loss of stimulus control and indiscriminate responding would also result in many responses during brief-stimulus presentations. However, very few responses occurred during brief-stimulus presentations, and *d*-amphetamine did not appreciably alter these rates.

Of interest is the difference in findings between Experiments 1 and 2. Only in the latter did the paired brief stimulus act consistently as a conditioned reinforcer, and only in the latter did the drug produce differential enhancing effects. Perhaps for *d*-amphetamine to enhance the effectiveness of a food-paired stimulus, that stimulus must already be serving as a conditioned reinforcer. This conclusion, however, seems to be at odds with the results reported by Files et al. (1989), who showed differential drug effects between paired brief-stimulus and no-stimulus conditions despite the fact that no difference was evident in nondrug performance (i.e., their brief stimulus did not act as a conditioned reinforcer when no drug was administered). When the results of Experiment 2 are compared to those of Files et al., a question presents itself. Should one label as a conditioned reinforcer a stimulus that is not demonstrated to be one by traditional behavioral criteria, but which seems to function as one only when a drug is administered? If so, then one may speak reasonably of the drug "revealing" the conditioned reinforcing effectiveness of the stimulus. If not, then new behavioral categories are needed, or one should speak of the drug as "establishing" the stimulus as a conditioned reinforcer. Our preference is not for revelations or for expanding the number of categories, but for the view that the drug in such cases increases the effectiveness of the pairing operation in establishing the stimulus as a conditioned reinforcer. Such an interpretation is consistent with findings that amphetamine accelerates the pace of respondent conditioning (e.g., Franks & Trouton, 1958); many theorists suggest that respondent conditioning underlies the phenomenon of conditioned reinforcement (e.g., Fantino, 1977; Mazur, 1990; Skinner, 1938). We suggest, tentatively, that stimulant drugs like methylphenidate and amphetamine, in circumstances like those used in the present experiments, have a behavioral mechanism of action (cf. T. Thompson & Schuster, 1968). Specifically, such drugs increase the effectiveness of pairing a stimulus with food (or other primary reinforcers) in establishing that stimulus as a reinforcer. Whether this speculation has merit will be determined by future research.

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